

Treatment of fetal supraventricular tachycardia with flecainide acetate after digoxin failure

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Transplacentally administered digoxin is the drug of choice for the treatment of fetal supraventricular tachycardia. We describe a case of fetal supraventricular tachycardia associated with fetal hydrops that did not respond to digoxin treatment because of a lack of transplacental passage. In contrast, flecainide acetate crossed the placenta and cured the fetus. The clinical implications of this new treatment are discussed. (AM J OBSTET GYNECOL 1991;165:630-1.)

Key words: Flecainide acetate, digoxin, fetal tachycardia

The treatment of fetal supraventricular tachycardia has been a difficult task. The most commonly used drug is digoxin alone or in combination with propranolol or verapamil. We describe a case of supraventricular tachycardia unresponsive to digoxin that was successfully treated with flecainide acetate.

Case report

A 22-year-old, gravida 1, para 0 woman was referred to the York Hospital antenatal testing unit at 31 weeks' gestation for fetal arrhythmia. Real-time ultrasound (Toshiba 270 a-sonolayer, Toshiba America Medical Systems, Tustin, Calif.) revealed mild hydramnios and fetal ascites. Fetal M-mode echocardiography revealed fetal supraventricular tachycardia at a rate of 240 beats/min with no evidence of atrioventricular block. Two-dimensional echocardiography revealed a structurally normal fetal heart with moderate dilatation. Duplex pulsed-wave and color Doppler ultrasonography revealed tricuspid regurgitation and reverse flow in the inferior vena cava. Myocardial contractility was decreased with a right ventricular fractional shortening consistently <20%. Treatment was initiated with digoxin transplacentally and 48 hours later the fetal heart rate (FHR) was 204 beats/min in spite of a maternal serum level of 2.1 ng/ml. Fetal umbilical vein level was 0.9 ng/ml (therapeutic, 0.9 to 2.0 ng/ml). Flecainide acetate was started at a dosage of 100 mg orally every 8 hours. Over the next 96 hours FHR converted to normal sinus rhythm of 120 beats/min. Maternal flecainide trough levels drawn at 24 hours, 48 hours, and 96 hours after initiation of treatment were 0.5, 0.6, and 0.8 µg/ml (therapeutic level, 0.2 to 1

µg/ml). No maternal side effects were noted and daily maternal electrocardiogram for 3 days was normal. At 56 hours after conversion a decision was made to discontinue both the digoxin and flecainide because of nonreactive nonstress test (NST) results, which gradually became reactive after discontinuation of flecainide.

Approximately 36 hours after discontinuation of treatment the FHR returned to pretreatment levels. Flecainide acetate 150 mg was given orally alone and 30 minutes later the FHR converted to normal. The patient received an additional 150 mg in 12 hours and every 12 hours thereafter. After 4 days at a stable heart rate of 120 beats/min with nonreactive NST results, flecainide was gradually decreased by 50 mg decrements to 50 mg every 12 hours, and over this period of time the NST results became reactive with normal beat-to-beat variability. Maternal serum flecainide trough level during this period was 0.2 µg/ml.

Right ventricular function improved, inferior vena cava flow pattern returned to normal, and cardiac dimensions became normal within the first 20 days. The placental thickness declined from 6.4 to 4.2 cm and fetal ascites resolved completely in 10 days.

The patient was delivered vaginally at 41 weeks' gestation. Maternal and fetal serum trough levels of flecainide at the time of delivery were 0.2 and 0.1 µg/ml, respectively. The Apgar scores were 9 and 10 at 1 and 5 minutes, respectively, and the venous blood gas values were normal. Birth weight was 3480 gm. Postnatal echocardiography was normal. At 48 hours after birth the neonate was started on prophylactic digoxin.

Comment

Digoxin has been the drug of choice and its safety is recognized. However, digoxin alone is not successful in all cases and this most likely is a result of poor transplacental passage.¹ Flecainide has been used successfully in the treatment of children with tachyarrhythmia resistant to conventional treatment and was found to

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be safe.² Flecainide treatment of fetal supraventricular tachycardia by intravenous administration to the mother resulted in conversion of the fetal arrhythmia to regular sinus rhythm in less than 10 minutes.⁵ In this case, toxic maternal serum levels of digoxin (2.1 ng/ml) resulted in a fetal serum level of 0.9 ng/ml, which is at the lowest end of the therapeutic range. The addition of propranolol was rejected because of the theoretic risk of intrauterine growth retardation and the addition of verapamil was rejected because of its association with fetal and neonatal deaths. Direct intramuscular fetal injections were considered unsafe in this patient. Flecainide acetate was then chosen because it has been shown to pass across the placenta rather easily with fetal levels as high as 86% of maternal serum levels.

Flecainide acetate in this case has been associated with nonreactive NST results and poor beat-to-beat variability, a dosage-related event that subsides sponta-

neously with the lowering of the dosage. Surveillance for fetal well-being was performed by means of biophysical profile when the beat-to-beat variability was poor and with twice-weekly NSTs when the beat-to-beat variability returned to normal. Fetal blood sampling for determination of digoxin level was instrumental in defining the reason for the failure of digoxin to control the tachycardia. We conclude that the use of flecainide acetate for the treatment of supraventricular tachycardia may be a safe and successful alternative for arrhythmias resistant to digoxin.

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